AN 1970:18865 CAPLUS
DN 72:18865
TI Estrogenic and antiestrogenic activities of a number of steroids in behavioral estrus and vaginal smear assays in the ewe
AU Lindsay, D. R.; Scaramuzzi, R. J.
CS Univ. Sydney, Sydney, Australia
SO Journal of Endocrinology (1969), 45(4), 549-55
CODEN: JOENAK; ISSN: 0022-0795
DT Journal
LA English
CC 4 (Hormones and Related Substances)
AB Fourteen synthetic steroids and androstenedione were examd. in ovariectomized ewes for estrogenic activity when administered alone and with estradiol benzoate by i.m. **injection**. None of the compds. investigated was active when administered alone, as assessed by the vaginal smear assay, and only androstenedione produced a behavioral response. Androstenedione had a min. effective dose of 8.8 mg but was less active when administered i.v. Several steroids acted as antiestrogens when injected with estradiol benzoate. Eight steroids inhibited the behavioral response and 4 the vaginal response. An additive response was found with androstenedione for behavioral response and with 17.beta.-ethyl-17-hydroxy-19-nor-4-androsten-3-one for vaginal response. Vaginal and behavioral responses were not necessarily related, and responses obtained in the ewe to particular steroids were not identical with those obtained in lab. animals by other workers using similar tests.
ST steroids estrogenic; estrogenic steroids; behavior steroids; antiestrogenic steroids
IT Estrogenic hormones
RL: BIOL (Biological study)
(and inhibitors, steroids as, assay techniques in relation to)
IT Estrus
(estrogenic activity of steroids detn. by induction of behavioral, vaginal smear assay in relation to)
IT Vagina
(estrogenic activity of steroids detn. by smear from, behavioral estrus in relation to)
IT Steroids, biological studies
RL: BIOL (Biological study)
(estrogenic and antiestrogenic activities of, in behavioral estrus and vaginal smear assays)
IT 17913-39-2
RL: BIOL (Biological study)
(behavioral estrus and vaginal estrogen response inhibition by)
IT 65-04-3
RL: BIOL (Biological study)
(behavioral estrus and vaginal response inhibition by)
IT 63-05-8
RL: BIOL (Biological study)
(behavioral estrus augmentation and vaginal estrogen response inhibition by)
IT 64-82-4 2061-45-2 2061-46-3 26624-16-8 26624-17-9
RL: BIOL (Biological study)
(behavioral estrus inhibition by)
IT 52-78-8
RL: BIOL (Biological study)
(vaginal estrogen response augmentation by)
IT **26624-15-7**
RL: BIOL (Biological study)
(vaginal estrogen response inhibition by)

AN 1966:440387 CAPLUS
DN 65:40387
OREF 65:7576b-e
TI Effects of androgens, estrogens, and corticoids on strontium kinetics in man
AU Eisenberg, Eugene
CS Univ. of California, San Francisco
SO J. Clin. Endocrinol. Metab. (1966), 26(5), 566-72
DT Journal
LA English
CC 58 (Hormones)
AB Kinetic studies were made in subjects after intravenous administration of 10 meq. Sr, before and during treatment with steroid hormones, to det. the effects of these agents on the bone deposition rate. Oral administration of fluoxymesterone, oxandrolone, oxymetholone, 7,17-dimethyltestosterone, and norethandrolone (10, 5, 7.5, 1.25, and 20 mg./day) or intravenous **injection** of testosterone enanthate, testosterone caprinoyl ace tate, or nandrolone phenopropionate (200, 200, and 50 mg., resp., every 2 weeks) decreased the urinary excretion rate of Sr, when administered for 6 weeks. Oral administration of conjugated equine estrogen, methallenestrol, ethynylestradiol, or 16.alpha.-methylestriol 16.beta.,17.beta.-3-methyl ether (2.5, 9, 0.1, and 20 mg./day, resp.) similarly decreased the urinary excretion rate and also decreased Sr deposition in bone by .apprx.0.6 l. of miscible pool/24 hrs.; since these were all patients with postmenopausal osteoporosis, this represented .apprx.10% decrease in the bone Sr deposition rate. The androgens and estrogens therefore appear to be anticatabolic for bone, and estrogens may also be antianabolic. Oral administration of cortisol, prednisone, triamcinolone, 6.alpha.fluorotriamcinolone, dexamethasone, or 6.alpha.-fluoroprednisolone (80-120, 20-30, 12-18, 24, 3, or 12 mg./day, resp.) did not decrease the bone deposition rate but did increase the urinary excretion rate of Sr; the corticoids therefore did not appear to be antianabolic for bone. The decrease in bone mass which eventually occurs following corticoid administration is probably the result of accelerated bone resorption. Correction of bone deposition rates of Sr for fecal excretion rates did not affect the results. The results did not show whether the changes in urinary excretion rates induced by both gonadal steroids or glucocorticoids were attributable to effects on the kidney, on bone, or on both. 38 references.
IT Bones
Urine
(strontium in, effect of androgens, corticosteroids and estrogens on)
IT Androgenic hormones or principles
Corticosteroids
Estrogenic hormones or principles
(strontium metabolism response to)
IT Testosterone, heptanoate, mixt. with testosterone propionate
(strontium metabolism response to)
IT 76-43-7, Androst-4-en-3-one, 9-fluoro-11.beta.,17.beta.-dihydroxy-17-methyl- 434-07-1, 5.alpha.-Androstan-3-one, 17.beta.-hydroxy-2-(hydroxymethylene)-17-methyl-
(in strontium metabolism)
IT 7440-24-6, Strontium
(metabolism of, effect of androgens, corticosteroids and estrogens on)
IT 53-34-9, Pregna-1,4-diene-3,20-dione, 6.alpha.-fluoro-11.beta.,17,21-trihydroxy-
(prepn. of)
IT 219-13-6, Cyclopenta[5,6]naphtho[1,2-c]pyran
(steroid derivs., strontium metabolism response to)
IT 50-02-2, Pregna-1,4-diene-3,20-dione, 9-fluoro-11.beta.,17,21-trihydroxy-16.alpha.-methyl- 50-23-7, Cortisol 53-03-2, Pregna-1,4-diene-3,11,20-trione, 17,21-dihydroxy- 124-94-7, Pregna-1,4-diene-3,20-dione,

9-fluoro-11.beta.,16.alpha.,17,21-tetrahydroxy- 807-38-5, Pregna-1,4-diene-3,20-dione, 6.alpha.,9-difluoro-11.beta.,16.alpha.,17,21-tetrahydroxy-
(strontium in urine in response to)

IT 52-78-8, 19-Nor-17.alpha.-pregn-4-en-3-one, 17-hydroxy- **53-39-4**, 2-Oxa-5.alpha.-androstan-3-one, 17.beta.-hydroxy-17-methyl- 57-63-6, 19-Nor-17.alpha.-pregna-1,3,5(10)-trien-20-yne-3,17-diol 62-90-8, Estr-4-en-3-one, 17.beta.-hydroxy-, hydrocinnamate 517-18-0, 2-Naphthalenepropionic acid, .beta.-ethyl-6-methoxy-.alpha.,.alpha.-dimethyl- 5108-94-1, Estra-1,3,5(10)-triene-16.beta.,17.beta.-diol, 3-methoxy-16-methyl- 5874-98-6, Testosterone, 3-oxododecanoate 10350-44-4, Androst-4-en-3-one, 17.beta.-hydroxy-7

AN 1969:93684 CAPLUS
DN 70:93684
TI Nutritional and metabolic effects of anabolic steroids and corticosteroids
AU Albanese, Anthony A.
CS Nutr. and Metab. Res. Div., Burke Rehabil. Center, White Plains, NY, USA
SO Journal of the American Medical Women's Association (1969), 24(1), 42-51
CODEN: JAMWAN; ISSN: 0091-7427
DT Journal
LA English
CC 4 (Hormones)
AB The steroid protein activity index (SPAI), a measurement of anabolic activity, was reported for orally administered anabolic steroids (testosterone propionate, 19-nortestosterone, norethandrolone, oxandrolone, 4-hydroxy-17.alpha.-methyltestosterone, methandrostenolone, stanozolol, norbolethione, 17.beta. - trimethylsiloxyandrost-4-en-3-one, BAS-71, and 17.beta.-hydroxy-2-oxa-19-norandrosta-4,9(10)-dien-3-one), corticosteroids (prednisone, prednisolone, triamcinolone, dexamethasone, paramethasone, betamethasone, and fluocortolone), as well as for parenteral anabolic steroids (dromostanolone propionate, stanozolol, methenolone enanthate, bolmantalate, oxandrolone, bolandiol dipropionate (SC-7525), SKF-6611, and SKF-8048). Trials with the oral administration of corticosteroids, followed by a period of combined corticosteroid and anabolic steroid **therapy**, permitted the detn. of the anticor-ticocatabolic activity index (ACAI). From this, the pos. action of the anabolic steroids on N retention could be quantitated and dosage relation established.
ST anabolic steroids activity; corticoids anabolic steroids; steroids anabolic corticoids; steroid protein activity index
IT Proteins
RL: BIOL (Biological study)
(metabolic retention of, detn. of steroid action on)
IT BAS 71
RL: BIOL (Biological study)
(nitrogen retention response to, calcn. of)
IT Cyclopenta[5,6]naphtho[1,2-c]pyran, oxasteroid derivs.
RL: SPN (Synthetic preparation); PREP (Preparation)
(prepn. of)
IT 50-02-2, biological studies 50-24-8, biological studies 52-78-8
53-03-2 53-33-8 **53-39-4** 57-85-2 72-63-9 124-94-7
145-12-0 152-97-6 302-96-5 303-42-4 378-44-9 434-22-0 521-12-0
797-58-0 1164-99-4 1491-81-2 1986-53-4 5055-42-5 20111-37-9
22467-98-7
RL: BIOL (Biological study)
(nitrogen retention response to, calcn. of)

AN 1986:45939 CAPLUS
DN 104:45939
TI The effect of androgens on the pulsatile release and the twenty-four-hour mean concentration of growth hormone in peripubertal males
AU Link, Kathleen; Blizzard, Robert M.; Evans, William S.; Kaiser, Donald L.; Parker, Mark W.; Rogol, Alan D.
CS Med. Cent., Univ. Virginia, Charlottesville, VA, 22908, USA
SO Journal of Clinical Endocrinology and Metabolism (1986), 62(1), 159-64
CODEN: JCEMAZ; ISSN: 0021-972X
DT Journal
LA English
CC 2-4 (Mammalian Hormones)
AB The effects of oxandrolone (Ox) [**53-39-4**] and testosterone (T) [58-22-0] on the mean concn. of growth hormone (GH) [9002-72-6], the pattern of GH secretion, and somatomedin C (SmC) [67763-96-6] concns. in boys with short stature and (or) delayed sexual development were studied to det. whether their growth-promoting effects might be mediated through endogenous GH release. Ten boys received Ox (0.1 mg/kg/day, orally) for 65 days, and 5 boys received T propionate (7.5 mg, i.m., for 7 days), followed by T enanthate (100 mg, i.m., monthly for 3 mo). Serum GH was measured in samples obtained at 20-min intervals for 24 h before and 65 days after the initiation of **therapy**. SmC levels were measured twice during the same 24-h period before and 65 days after initiation of **therapy**. In the boys treated with T, there were increases in the mean concn. of GH (4.3-fold), in the no. of GH pulses .gtoreq.10 ng/mL, (1.6 vs. 4.8/24 h), and in the SmC levels (0.82 vs. 2.3 .mu./mL). There were, however, no significant changes in the boys treated with Ox. Both Ox and T improved the growth rates; however, T increased the growth rate by 0.95 cm/mo, and Ox increased the growth rate by 0.24 cm/mo. Thus, T, but not Ox, at the doses tested increases GH secretion in boys with short stature and (or) delayed sexual development. This increase in GH secretion may contribute to the increased growth rate in males at puberty.
ST androgen somatotropin secretion puberty; testosterone somatotropin secretion puberty; oxandrolone somatotropin secretion puberty; growth hormone secretion androgen
IT Blood serum
(growth hormone and somatomedin C of, of boy in puberty, androgens effect on)
IT Androgens
RL: BIOL (Biological study)
(growth hormone secretion response to, in puberty in boy)
IT Puberty
(male, growth hormone secretion in, in boy, androgens effect on)
IT **53-39-4** 58-22-0
RL: BIOL (Biological study)
(growth hormone secretion response to, in puberty in boy)
IT 67763-96-6
RL: BIOL (Biological study)
(of blood serum, of boy in puberty, androgens effect on)
IT 9002-72-6
RL: BIOL (Biological study)
(secretion of, by boy in puberty, androgens effect on)